Effect of dietary sources of calcium and protein on hip fractures and falls in older adults in residential care: cluster randomised controlled trial

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ABSTRACT

OBJECTIVE

To assess the antifracture efficacy and safety of a nutritional intervention in institutionalised older adults replete in vitamin D but with mean intakes of 600 mg/day calcium and <1 g/kg body weight protein/day.

DESIGN

Two year cluster randomised controlled trial.

SETTING

60 accredited residential aged care facilities in Australia housing predominantly ambulant residents.

PARTICIPANTS

7195 permanent residents (4920 (68%) female; mean age 86.0 (SD 8.2) years).

INTERVENTION

Facilities were stratified by location and organisation, with 30 facilities randomised to provide residents with additional milk, yoghurt, and cheese that contained 562 (166) mg/day calcium and 12 (6) g/day protein achieving a total intake of 1142 (353) mg calcium/day and 69 (15) g/day protein (1.1 g/kg body weight). The 30 control facilities maintained their usual menus, with residents consuming 700 (247) mg/day calcium and 58 (14) g/day protein (0.9 g/kg body weight).

MAIN OUTCOME MEASURES

Group differences in incidence of fractures, falls, and all cause mortality.

RESULTS

Data from 27 intervention facilities and 29 control facilities were analysed. A total of 324 fractures (135 hip fractures), 4302 falls, and 1974 deaths were observed. The intervention was associated with risk reductions of 33% for all fractures (121 *v* 203; hazard ratio 0.67, 95% confidence interval 0.48 to 0.93; P=0.02), 46% for hip fractures (42 *v* 93; 0.54, 0.35 to 0.83; P=0.005), and 11% for falls (1879 *v* 2423; 0.89, 0.78 to 0.98; P=0.04). The risk reduction for hip fractures and falls achieved significance at five months (P=0.02) and three months (P=0.004), respectively. Mortality was unchanged (900 *v* 1074; hazard ratio 1.01, 0.43 to 3.08).

CONCLUSIONS

Improving calcium and protein intakes by using dairy foods is a readily accessible intervention that reduces the risk of falls and fractures commonly occurring in aged care residents.

TRIAL REGISTRATION

Australian New Zealand Clinical Trials Registry ACTRN12613000228785.

WHAT IS ALREADY KNOWN ON THIS TOPIC

Few studies have investigated the efficacy and safety of a nutritional approach to reduction of fracture risk in institutionalised older adults

One study using pharmacological doses of calcium and vitamin D reduced hip fractures in female nursing home residents with low calcium intakes and vitamin D deficiency

WHAT THIS STUDY ADDS

Supplementation using high calcium, high protein dairy foods reduced falls and fractures in vitamin D replete older adults in aged care

Introduction

Longevity increases the proportion of older adults in the population. The accompanying increased prevalences of chronic illnesses, loss of musculoskeletal mass, frailty, and bone fragility increase the risk of falls and fractures.[1] Loss of independence increases the number of people needing full time institutionalised care, the source of around 30% of all hip fractures in the community.[2] Thus, targeting an intervention to all aged care residents is a rational approach to reducing the fracture burden in the whole community.

The widespread use of antiresorptive therapy is unlikely to reduce this fracture burden because of a paucity of evidence of antifracture efficacy in people over 80 years of age, the common occurrence of adverse events, and high cost given the large numbers of people that must be treated.[3] However, these people often have calcium intakes below 700 mg daily, an amount unlikely to offset obligatory loss of calcium.[4] They also often have protein intakes below 1 g/kg body weight/day, predisposing to loss of lean muscle mass.[5] Thus, an alternative approach is to target all institutionalised older adults with a non-pharmaceutical nutritional intervention.

Few studies have investigated the efficacy and safety of a nutritional approach to reduction of fracture risk in aged care residents. Chapuy and colleagues showed antifracture efficacy with pharmacological doses of calcium and vitamin D in female nursing home residents with low calcium intakes and vitamin D deficiency.[6] No studies have examined the effects of protein supplementation on reduction of fracture risk, despite evidence of improved muscle function and reduced falls.[7]

Consumption of milk, yoghurt, and cheese, foods rich in calcium and protein, slows bone loss and improves insulin-like growth factor 1.[8 9] These foods are widely available, palatable, and low cost and so are likely to be adhered to. Accordingly, we conducted a prospective,

two year, cluster randomised controlled trial to test the hypothesis that achieving recommended intakes of 1300 mg/day calcium and 1 g protein/kg body weight will reduce the risk of fragility fractures and falls when targeted to institutionalised older adults replete in vitamin D but with intakes below these levels.

Methods

Study design

This two year, cluster randomised controlled trial involved recruitment of 60 residential aged care facilities housing 7195 older adults in metropolitan Melbourne and regional Victoria, Australia, between December 2013 and August 2016. To ensure similar standards of care, we recruited only facilities accredited with the Australian Aged-Care Accreditation Agency that housed predominantly ambulant residents. These facilities are similar to residential care in the UK and assisted care facilities in the US. Facilities recruited were representative of charitable, private, and religious organisations, with an even distribution of small (≤50 beds), medium (51-100 beds), and large (>100 beds) facilities (supplementary figure C). The ratio of women to men and the age of residents were representative of the national average.[10]

Inclusion criteria

Randomisation was by facility, not by individuals. For inclusion, facilities were required to provide no more than two servings of dairy foods daily, which was assessed from menu audits, as this level of provision is associated with dietary intakes of <1 g/kg body weight and 600 mg calcium daily.[11] Vitamin D adequacy is maintained in residents through routine supplementation as foods are not fortified with vitamin D. We included only permanent residents in data analyses—that is, we excluded data from respite residents.

Randomisation procedure

The unit of randomisation was facility, as the intervention was delivered to all residents by the food service at each facility. Eligible facilities were randomly assigned in a 1:1 ratio to either intervention (n=30) or control (n=30), with the control facilities maintaining their existing menus. The randomisation was done with the use of a computer, with block sizes being varied according to organisation (to ensure similar procedures and policies), and was stratified by geographical location (to ensure similar socioeconomic status). A statistician independent of the study did the randomisation and provided the concealed group allocation to the principal investigator (SI) who, in turn, conveyed this allocation to the facility. SI was not involved in any data collection. An organisation may have between two and 10 facilities, and randomisation was done within an organisation.

Consent

Facility managers consented to provide de-identified details of age and sex of residents, as well as access to all incident reports including those for falls and fractures. Reporting of all incidents of any nature is a mandatory requirement of all accredited aged care providers. The accreditation agency regularly audits incident reports. Facilities are sanctioned if breaches are observed, with potential for accreditation to be revoked and government funding terminated. Falls (time, location, circumstances, and outcome/injury), fractures, and other adverse events were verified from these incident reports. These reports were maintained at all facilities. An independent medically trained person blinded to study allocation verified fractures by using hospital radiographs and radiographic reports. Residents and families were informed of the study during regular meetings. A subset of 371 residents from all facilities voluntarily consented to have dietary intake recorded, medical records reviewed, blood sampling, and measurement of body composition, bone mineral density, and bone microarchitecture performed. A next of kin consented for an additional 345 residents to allow dietary intake to be recorded and medical records reviewed.

Intervention

We classified dairy foods by using the Australian Dietary Guidelines, with a "serving" defined as 250 mL of milk, 200 g of yoghurt, and 40 g of cheese.[12] Lactose-free options were provided to accommodate the few participants (<0.001%) with reported lactose intolerance. Butter, cream, and ice cream were not provided, as they contain little calcium or protein. All facilities prepared and cooked foods on site. We assigned intervention facilities a food service dietician to assist food service staff to increase the provision of dairy foods at all meals and snacks. Methods used to increase dairy foods included use of milk powder to fortify milk used in recipes and beverages. Dairy based desserts and snacks were offered in place of less nutritious foods such as cakes and biscuits. Foods provided were based on the preferences expressed by the residents at intervention sites.

Dairy foods were provided in-kind by Fonterra International (New Zealand) and distributed by a commercial food distribution company not associated with the project (Bidfoods, Australia). Use of a single distributor ensured accurate recording of costs for all dairy foods provided, with invoices used to verify compliance data. During dietary assessments, foods and beverages were weighed on a food scale (±1 g) (Sohnele Page Profi, Germany) at all facilities. During two days every three months, dieticians assessed compliance by using the validated visual estimation of plate waste, with data collected from 55 000 foods and beverages during the study.[13] We used nutritional analysis software (FoodWorks, Australia) or the Australian food composition database NUTTAB 2010 to calculate nutrient intakes.

Data monitoring

Data safety monitoring was carried out by the Study Trial Review Board, which was provided with quarterly reports.

Outcomes

As per the approved study protocol, all pre-specified primary and secondary outcomes have been reported. The primary outcome was time to fragility fracture. Secondary outcomes were time to fall and changes in bone morphology and biochemistry. The tertiary outcomes of all cause mortality and changes in body composition are also reported. Exploratory outcomes including quality of life and muscle function were not examined (see original and final study protocols). Fasting morning serum samples were obtained from 189 residents at baseline and 12 months for measurement of 25-hydroxy-vitamin D (baseline only), C terminal telopeptide of type 1 collagen (a measure of bone resorption), procollagen type 1 N propeptide (a measure of bone formation), parathyroid hormone (Roche Cobas E170), and insulin-like growth factor 1 (LIASON) (supplementary figure A).

Body composition and bone morphology were assessed at baseline and 12 months in 72 residents (supplementary figure B). Total and appendicular (arms and legs) lean mass and fat mass were determined from total body scans, and bone mineral density was measured at the lumbar spine and femoral neck using dual x-ray absorptiometry (Prodigy, GE Lunar, Madison, WI, CV=1%). Volumetric bone mineral density (the amount of bone contained within the external volume of bone, in g/cm^3) was measured at the distal tibia and distal radius by using high resolution peripheral quantitative computed tomography (Scanco Medical AG, Switzerland, CV 0.5–4.0%).[14] Cortical porosity was determined using automated image processing (StrAx1.0, Straxcorp, Melbourne, Australia).

Blinding and sample size

Once a facility was randomised, only the principal investigator, food service research dieticians, facility managers, and food service staff were aware of the allocation. Data acquisition and analyses were carried out by staff blinded to group allocation (SP, XW, MB, AGZ, and TN). Residents were blinded to the study; permission to conduct the study was obtained from the aged-care provider and facility managers. Some of the intervention strategies were not visible—for example, fortification of milk with milk powder or modification of recipes. Some residents may have been aware of some changes, such as provision of cheese and biscuits for snacks, but not the reason for the changes.

The sample size was determined on the basis of a hypothesised effect size and intra-cluster correlation coefficient (r). Under the hypothesis that the intervention reduces the risk of fracture by 30%, based on previous antifracture calcium/vitamin D intervention in this setting, and that r ranges from 0.10 to 0.50, the sample size needed was 25 to 50 residents per facility and 25 facilities per arm to achieve the power of 80%.[6] From falls data, we used an r of 0.20 to calculate the sample size.[15] To account for approximately 20% annual attrition, we recruited 60 facilities with a minimum of 50 residents per facility.

At the start of the study, 3980 permanent residents were living in the participating facilities. We refer to these residents as the inception cohort. Recruitment continued throughout the 24 months to ensure that the required sample size was maintained, so we included data from residents admitted to facilities that replaced initial residents lost to follow-up due to death or discharge in analyses. We refer to these residents as the replacement cohort. In total, an additional 3215 residents were admitted to facilities after the study had started. We obtained details of new residents and those lost to follow-up from admission and discharge records from each facility.

Analyses

We expressed baseline data as mean and standard deviation, with the unit of analysis being clusters. We expressed fracture incidence, falls, and deaths per 100 person years of follow-up. We used the product limit (Kaplan-Meier) method to determine the cumulative risk of an event. No data were missing for these primary and secondary outcomes. The duration of follow-up was based on date of study entry to date of an event. When no event occurred, duration of follow-up was date of study entry to date of study termination.

As individuals were "nested" within clusters (facilities), the primary analysis was based on the mixed effects Cox's proportional hazards model; effects of intervention, age, and sex were fixed effects, and the facility was considered the random effect (see supplementary methods for additional statistical analysis). We expressed the results as a hazard ratio with 95% confidence limits. We used the "coxme" package to estimate model parameters. We also used the Fine-Gray sub-distribution method with the "cmprsk" package to do mortality competing risk analysis.

We tested between group differences in serum biomarkers and measurements of body composition and bone morphology at baseline with the weighted t test, with cluster being the unit of analysis. Biomarkers were log transformed if they were not normally distributed. We analysed effects of the intervention by using the mixed effects model in which the within person change in outcome was modelled as a function of treatment or control group, time of follow-up, age, weight, and sex. All analyses used the R Statistical Environment.

Amendments to protocol and statistical analysis plan

Initially, facilities were matched only by location to account for socioeconomic status. We also accounted for organisations, as they contributed varying numbers of facilities and had different policies and procedures. We included facilities providing less than two servings of dairy food daily, as dietary assessments for all residents was not feasible. We quantified two day instead of three day diets, as this was adequate to capture regular intakes.[11] We did not assess osteocalcin, as sufficient information is obtained from C terminal telopeptide of

type 1 collagen and procollagen type 1 N propeptide. We used only all cause mortality as a tertiary outcome, as cardiovascular events were not obtainable and causes of death were poorly documented. We did not examine exploratory outcomes related to quality of life and muscle function, as unanticipated attrition reduced the sample size resulting in insufficient power to detect an effect of treatment. We did not include bayesian analyses and imputations, as no values were missing for falls and fracture outcomes (original and final study protocols).

Patient and public involvement

We consulted aged care residents, providers, and food service staff after the initial feasibility study that guided the design of this intervention.[16] The manuscript was read by non-academics.

Results

Of the 60 facilities, 54 completed the 24 month intervention (fig 1). One control facility and three intervention facilities withdrew after randomisation. Two intervention facilities closed at months 15 and 20, but we included the data up to the date of closure. At baseline, the two groups had comparable demographics and were vitamin D replete. Daily baseline calcium and protein intakes were 689 (SD 266) mg and 57 (16) g respectively (table 1; supplementary figure D).

Nutritional changes

Dairy food intake increased from 2.0 to 3.5 servings daily in the intervention facilities (fig 2). The additional dairy foods, equivalent to 250 mL of milk plus 20 g cheese or 100 g yoghurt, provided 562 (166) mg calcium, achieving 1142 (353) mg calcium daily, and 12 (6) g protein, achieving an intake of 69 (15) g (1.1 g/kg body weight) daily. In control facilities, residents' dairy intakes remained at less than two servings daily providing 700 (247) mg calcium and 58 (14) g protein (0.9 g/kg body weight) daily. No adverse gastrointestinal events related to the intervention were reported. No detectable within or between group differences in energy intake were observed during follow-up (fig 2). However, we observed group differences for the change in body weight (table 2). In absolute terms, no weight change occurred in the intervention group (0.3 (95% confidence interval −0.8 to 1.4) kg; P=0.56). In controls, a weight loss of 1.4 (0.6 to 2.1) kg, (P<0.001) was due to a 0.3 (−0.6 to 0.0) kg decline in appendicular lean mass (P=0.03) and 0.8 kg (−1.6 to −0.2) decline in total body fat mass (P=0.02).

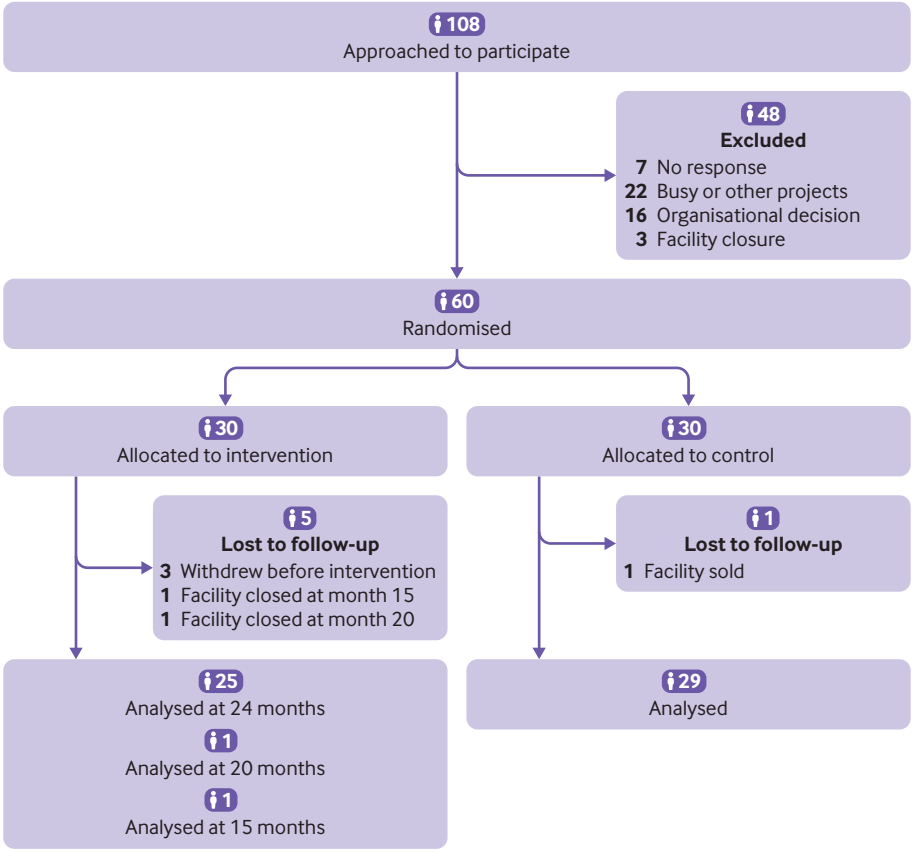

Fig 1 | Flowchart for participating aged care facilities. Of 108 eligible facilities, 48 were excluded, leaving 60 randomised to intervention or control. Three intervention facilities and one control facility did not start study, leaving 27 intervention and 29 control facilities participating, of which two intervention facilities closed during study period. Median number of residents in intervention and control groups were 111 (interquartile range 75-147) and 125 (88-163), respectively; P=0.42 by Wilcoxon's rank test

Table 1 | Baseline characteristics of residents in clusters assigned to two years of dairy supplementation (intervention) or maintenance of existing menu (controls). Values are mean (standard deviation) unless stated otherwise

Characteristics	Intervention (n=3301)	Controls (n=3894)
No of clusters (facilities)	27	29
Median No of residents per cluster	111	125
No (%) female	2311 (70)	2609 (67)
Age, years	86 (2.3)	86 (2.2)
Height (m)	1.60 (0.02)	1.61 (0.03)
Weight (kg)	66 (6.3)	68 (8.4)
Median (IQR) medications*	11 (8-14)	12 (9-16)
Median (IQR) medical conditions*	9 (7-12)	10 (7-14)
No (%) with previous fracture*	184 (41)	183 (37)
No (%) with cognitive impairment*	189 (52)	237 (53)
No (%) with cardiovascular disease*	301 (66)	309 (63)
No (%) malnourished, at risk, normal†	70 (17), 272 (66), 70 (17)	25 (11), 158 (66), 55 (23)
Biochemistry	**(n=170)**	**(n=135)**
25-hydroxy-vitamin D, nmol/L	72 (15)	72 (21)
C terminal telopeptide of type 1 collagen, ng/mL	0.41 (0.14)	0.39 (0.11)
Procollagen type 1 N propeptide, µg/L	52.9 (19.1)	48.5 (9.7)
Parathyroid hormone, pg/mL	6.85 (2.04)	7.27 (1.74)
Insulin-like growth factor 1, nmol/L	15.4 (2.8)	15.1 (3.1)
Body composition	**(n=77)**	**(n=79)**
Total lean mass, kg	39.7 (8.6)	39.8 (8.1)
Appendicular lean mass, kg	16.3 (1.2)	16.4 (3.7)
Fat mass, kg	22.5 (9.5)	27.2 (11.0)
Bone morphology	**(n=77)**	**(n=79)**
Femoral neck BMD, g/cm^2	0.76 (0.08)	0.74 (0.12)
Lumbar spine BMD, g/cm^2	1.08 (0.22)	1.13 (0.23)
Distal tibia:		
Total volumetric BMD, mgHA/cm^3	215 (44)	228 (63)
Cortical porosity, %	75 (5.6)	75 (6.6)
Trabecular volumetric BMD, mgHA/cm^3	155 (35)	167 (47)
Distal radius:		
Total volumetric BMD, mgHA/cm^3	257 (41)	247 (68)
Cortical porosity, %	68 (3.8)	69 (7.5)
Trabecular volumetric BMD, mgHA/cm^3	155 (34)	148 (47)

BMD=bone mineral density; IQR=interquartile range.
*n=457 intervention; n=494 controls.
†Mini Nutrition Assessment Score: 24-30=normal nutritional status; 17- 23.5=at risk of malnutrition; <17=malnourished (n=412 intervention; n=238 controls).

Fractures, falls, and mortality

During 90 557 person months of follow-up (mean 12.6 (8.9) months), 324 fractures occurred: 121 (3.7%) in the intervention group and 203 (5.2%) in controls—a 33% risk reduction (hazard ratio 0.67, 95% confidence interval 0.48 to 0.93; P=0.02). Post hoc analysis indicated that the incidence of hip fracture was 1.3% (n=42) in the intervention group and 2.4% (n=93) in controls—a 46% risk reduction (hazard ratio 0.54, 0.35 to 0.83; P=0.005). The separation in cumulative incidence of fractures between the groups achieved significance at five months for all fractures (P=0.02) and hip fractures (P=0.02) (fig 3). Competing risk analysis adjusted for mortality showed that the intervention was associated with average reductions in fracture risk of 27% (hazard ratio 0.73, 0.58 to 0.92) for all fractures and 44% (0.56, 0.39 to 0.82) for hip fractures.

The cumulative incidence of falls was 57% (n=1879) in the intervention group and 62% (n=2423) in controls—an 11% relative risk reduction (hazard ratio 0.89, 0.78 to 0.98; P=0.04). The separation in the incidence of falls between groups achieved significance at three months (P=0.04) (fig 3). All but one fracture was the result of a fall. Mortality did not differ between the intervention and control groups (27% (n=900) *v* 28% (n=1074), respectively; hazard ratio 1.01, 0.43 to 3.08; P=0.91) (fig 3). The numbers needing treatment to prevent any fracture, hip fracture, or a fall were 52, 82, and 17, respectively.

To explore the veracity of the observations made in the entire cohort (n=7195), we examined the effects of the intervention on fracture risk and falls relative to controls in a post hoc analyses of residents present at the start of the study (n=3980, the inception cohort) and residents added after its start (n=3215, the replacement cohort), separately. The inception cohort was older than the replacement cohort (mean 86.5 (8.1) *v* 85.2 (8.4) years; P<0.001). However, age did not differ between the intervention and control groups in either the inception cohort (mean 86.7 (8.2) *v* 86.4 (8.0) years, respectively; P=0.25) or the replacement cohort (85.1 (8.5) *v* 85.3 (8.2) years, respectively; P=0.601). We observed significant reductions in all fractures, hip fractures, and falls in the intervention group relative to the controls in both the inception and replacement cohorts (see supplementary figure E).

Biochemistry and bone morphology

The subgroup providing data for biochemistry, body composition, and bone morphology did not differ from

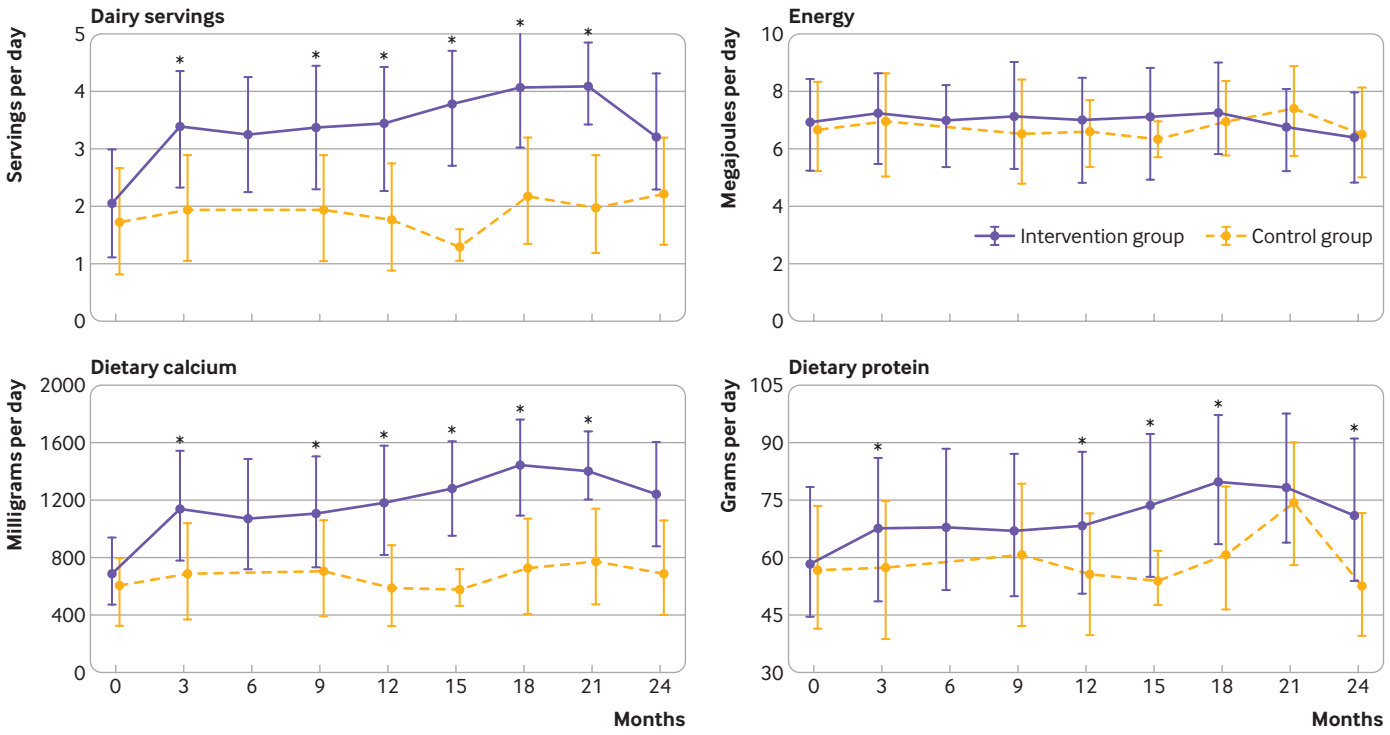

Fig 2 | Mean (SD) daily dietary intake of dairy servings, energy, calcium, and protein at baseline (regular menu) and during two year study period in intervention group compared with controls. *P<0.05 denotes significant difference between groups at corresponding time point

the entire cohort in age (mean 85.9 (8.2) *v* 85.6 (8.2) years), proportion of women to men (70% (n=74) *v* 69% (n=57)), and proportion with previous fractures (38% (n=40) *v* 39 (n=32)). As shown in table 2, at 12 months, we observed a 20.4% between group difference in serum C terminal telopeptide of type 1 collagen (P=0.002), the result of no change in the intervention group and a 13.1% increase in controls (P<0.05). We observed no between group difference in procollagen type 1 N propeptide or parathyroid hormone, but we observed a 7.9% between group difference in serum insulin-like growth factor 1 (P=0.04), the result of a 5.9% increase in the intervention group (P<0.05) and no change in controls.

We observed a 1.8% between group difference in spine bone mineral density (P=0.04), the result of a

Table 2 | Mean (standard deviation) percentage change from baseline at month 12 in biochemistry and bone morphology in each group and between group mean percentage difference with 95% confidence interval.

	Intervention	Control	Mean between group difference (95% CI)	P value
Biochemistry	(n=106)	(n=83)		
Bone resorption marker (CTX)	−7.3 (40.7)	13.1 (45.5)*	−20.4 (−33.2 to −7.6)	0.002
Bone formation marker (P1NP)	−4.7 (35.4)	−3.9 (55.1)	−0.8 (−14.6 to 12.9)	0.90
Parathyroid hormone	1.1 (20.7)	−0.16 (32.0)	1.3 (−6.7 to 9.2)	0.76
Insulin-like growth factor-1	5.9 (27.0)*	−2.0 (22.4)	7.9 (15.7 to 0.2)	0.04
Bone morphology	(n=33)	(n=39)		
Lumbar spine BMD	2.1 (2.7)**	0.3 (2.4)	1.8 (0.1 to 3.5)	0.04
Femoral neck BMD	0.7 (3.0)	−1.0 (4.4)	1.7 (−0.3 to 3.7)	0.09
Distal radius:				
Total volumetric BMD	0.7 (2.7)	−2.6 (5.6)*	3.3 (0.6 to 6.0)	0.02
Trabecular volumetric BMD	0.9 (2.3)	−3.5 (8.7)	4.6 (0.4 to 8.2)	0.03
Cortical porosity	0.0 (1.7)	0.7 (3.3)	−0.6 (−2.3 to 1.0)	0.43
Distal tibia:				
Total volumetric BMD	−0.1 (2.5)	−2.1 (4.4)*	2.0 (−0.1 to 4.2)	0.07
Trabecular volumetric BMD	0.2 (2.3)	−2.2 (8.5)	2.4 (−1.4 to 6.2)	0.21
Cortical porosity	0.4 (1.3)	0.7 (1.2)*	−0.3 (−1.1 to 0.4)	0.39
Body composition	(n=33)	(n=39)		
Body weight	0.6 (8.3)	−1.9 (5.2)**	2.5 (0.6 to 4.1)	0.009
Lean mass	−0.2 (2.7)	−0.4 (2.8)	0.3 (−0.9 to 1.6)	0.60
Appendicular lean mass	0.3 (4.5)	−1.7 (5.0)*	2.0 (0.02 to 4.1)	0.05
Fat mass	3.8 (20.6)	−3.3 (8.1)*	7.3 (0.1 to 14.5)	0.05

BMD=bone mineral density; CTX=C terminal telopeptide of type 1 collagen; P1NP=procollagen type 1 N propeptide.
*P<0.05 for difference from baseline within group.
**P<0.001 for difference from baseline within group.

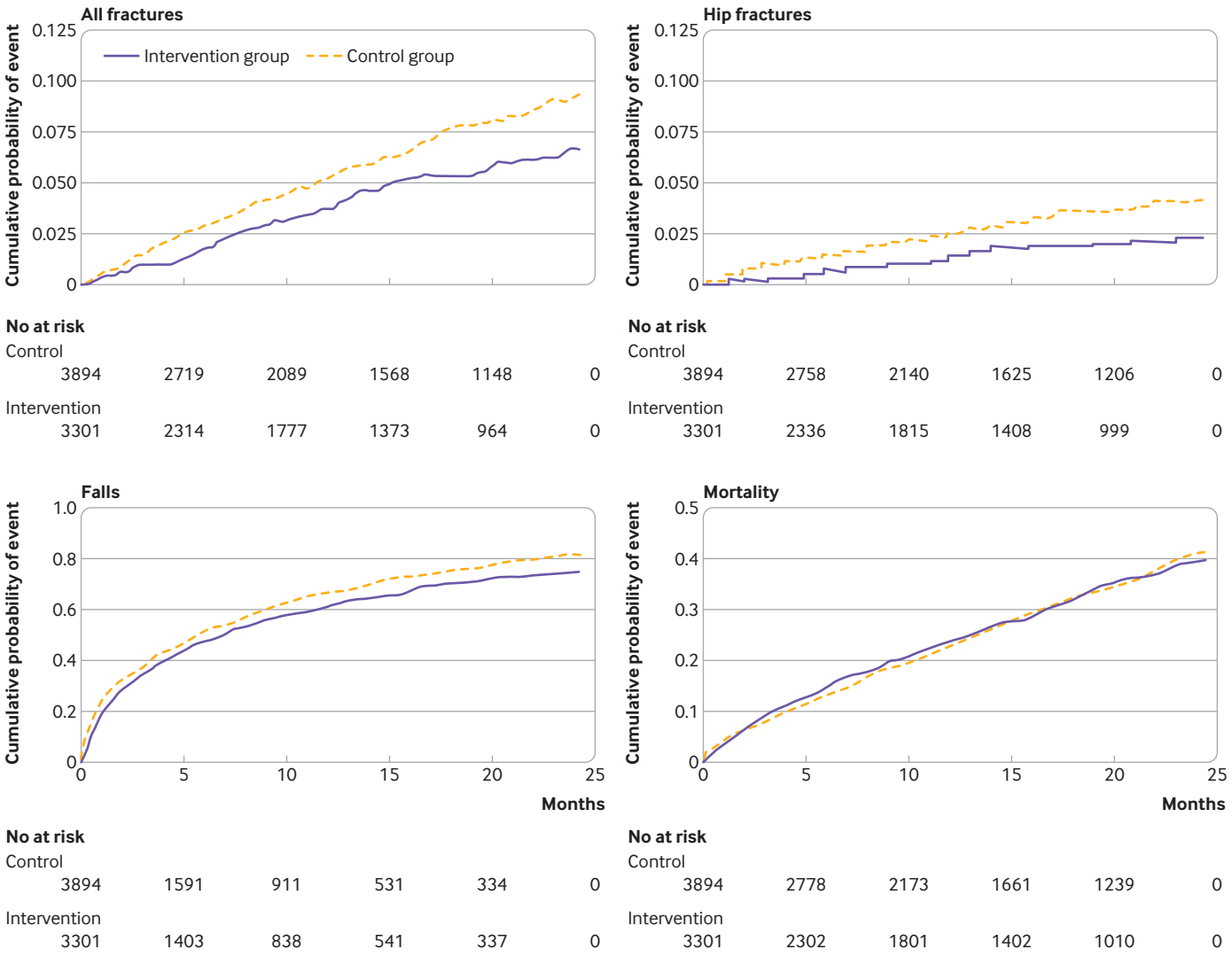

Fig 3 | Cumulative probability of event over 24 months for all fractures, hip fractures, falls, and mortality in intervention group and controls. Numbers of participants in two groups at risk for each event are shown below panels

2.1% increase in the intervention group (P<0.001) and no change in controls. The 1.7% between group difference in femoral neck bone mineral density was not significant (P=0.09). The 3.3% between group difference in distal radius total volumetric bone mineral density (P=0.02) and 2.0% between group difference in distal tibial total volumetric bone mineral density (P=0.07) were the result of decreases at each site in controls (both P<0.05). We observed a 4.6% between group difference in distal radial trabecular volumetric bone mineral density (P=0.03) due to a non-significant decrease in controls and a 0.7% increase in distal tibia cortical porosity in controls (P<0.05).

Discussion

This nutritional approach using high calcium and high protein dairy foods to increase calcium and protein intakes in institutionalised older adults replete in vitamin D was associated with a 33% reduction in risk of fractures of any type, a 46% reduction in risk of hip fractures, and an 11% reduction in risk of falls relative to controls. We found no group difference in all cause mortality.

Most interventions aimed at reducing fracture risk target a drug therapy to people with osteoporosis because they are at high risk of fracture. This approach confers a large benefit to the individual and does so cost effectively, because few people need to be treated to avert one event. However, averting fractures in small numbers of people at high risk does not reduce the burden of fractures in the community.

The population burden of fractures—the number of events, morbidity, mortality, and cost to the community—arises from the vast numbers of people with risk factors that confer a modest attributable risk to the individual.[17] For example, most fragility fractures in the community arise among women with osteopenia (bone mineral density T score −2.5 to −1 SD) because they form the largest segment of the community.[18] Likewise, most fractures attributable to nutritional inadequacy arise among the great many people with intakes of calcium and protein that are below recommended levels.[19] This nutritional inadequacy confers a small attributable risk to the individual but accounts for a large attributable fraction

of the fracture burden in the community as a whole. This is the Geoffrey Rose prevention paradox—a community based approach producing a small benefit to an individual may still confer a large benefit to the community.[17] Safety is essential because most individuals treated may derive little or no benefit from the intervention. For example, the Dietary Approach to Stop Hypertension study reduced blood pressure by replacing a "western" diet with a diet rich in fruit, vegetables, and low fat dairy foods—an approach associated with fewer cardiovascular events.[20]

Comparison with other studies

Most nutrition based studies assessing antifracture efficacy in aged care residents and people in the community used pharmacological doses of calcium with or without vitamin D.[21] In a meta-analysis of 17 of these studies, only two studies reported a reduction in fracture risk—a study of nursing home residents with calcium intakes <600 mg/day and vitamin D concentrations <50 nmol/L and a community based study in women and men ≥65 years of age with mean calcium intakes of 700 mg/day.[21] In the remaining 15 studies, poor compliance, large numbers of dropouts, and a low prevalence of the risk factor may have contributed to the null findings.[22 23] Benefits are unlikely if the prevalence of a risk factor (such as inadequate calcium and protein intakes) is low.[24] For example, in the meta-analysis, reduction in fracture risk reported with treatment was confined to the 7272 individuals with calcium intakes <700 mg/day (risk ratio 0.80, 95% confidence interval 0.71 to 0.89), not the 45 241 individuals with calcium intakes above 700 mg daily.[21] The reduction in fracture risk observed with calcium and protein rich foods in this study may have been the result of attention to several of the above factors. Compliance was optimised by supervised provision and consumption of the foods. Participants lost to follow-up were replaced by newly admitted residents. We intentionally targeted a cohort at high risk for fracture in whom low calcium and protein intakes were common and so were likely to partly contribute to the already high fracture burden in this community.

Mechanisms of fracture risk reduction

This nutritional intervention produced two unanticipated novel observations. The risk reduction for falls and fractures was detected by three and five months, respectively, and the relative risk reduction for fractures was similar to that found in trials using potent antiresorptive therapy to treat people at high risk due to osteoporosis. The two most likely explanations for each of these observations is a risk reduction for falls and slowing progression of bone fragility. Insulin-like growth factor 1 increased in the intervention group only, whereas the decrease in appendicular lean mass was confined to controls, consistent with the notion that protein intakes of 1-1.5 g/kg/day is needed to prevent protein catabolism and preserve or increase muscle mass in older adults, particularly those at risk of malnutrition or frailty.[25] The increase in serum C terminal telopeptide of type 1 collagen and deterioration in tibial and radial total volumetric bone mineral density in controls was not seen in the intervention group, consistent with slowing of bone loss and slowing of microstructural deterioration.[26] These changes were modest, but slowing microstructural deterioration disproportionately reduces progression of bone fragility because fragility increases as a power function to the bone loss producing it.[27]

Mortality did not differ between the groups. Some, but not all, studies suggest that milk consumption is associated with increased mortality but consumption of yoghurt and cheese (fermented foods) with reduced mortality and favourable blood lipid profiles.[28-30] Fermented and non-fermented dairy foods were used during the intervention. Milk consumption did not differ between the intervention and control groups (data not shown).

Limitations of study

The study has several limitations. Less than half of the participants had follow-up longer than 15 months. However, the reduction in risk of fractures and falls was detected within six months. Measures of dietary intakes and causes of secondary osteoporosis were obtained from the subgroup of 716 consented participants, not all 7195 residents, so compliance was monitored in about 10% of residents. However, recorded intakes of 55 000 foods and beverages are likely to be representative of all residents as most, if not all, food is provided by facilities. Assessment of body composition, bone morphology, and biochemistry was confined to a subgroup of residents. Attrition of these participants limited the power to examine differences in body composition, bone morphology, and biochemistry between the groups. Therefore, our ability to make inferences concerning the role of this intervention in slowing microstructural deterioration and loss of muscle mass is limited. Serum parathyroid hormone remained unchanged, perhaps owing to administration of around 1100 mg of calcium throughout the day as food, not as a single supplement of elemental calcium.[31] Moreover, this intervention used whole dairy foods, so any potential benefit of other components of the dairy matrix cannot be determined.

Summary and conclusions

In summary, ageing of the population is associated with a greater number of older adults needing full time institutionalised care. These individuals are often malnourished.[5] Although the risk of fracture attributable to undernutrition may be small in an individual, the large number of older adults in aged care confers a large fracture burden in the community; institutionalised people are the source of about 30% of all hip fractures.[2 17] A high calcium and high protein nutritional intervention reduced the risk of falls and fractures. This intervention was tailored to the preferences of the residents and was successfully

delivered through the food service using regular retail milk, yoghurt, and cheese incorporated into existing menus. In conclusion, this nutritional intervention has widespread implications as a public health measure for fracture prevention in the aged care setting and potentially in the wider community.

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Contributors: SI was involved in the conception, design, planning, and management of the study, data acquisition, interpretation of results, drafting the manuscript, and critically reviewing or revising the manuscript for important intellectual content. SP and JR were involved in data acquisition and management and critically reviewing or revising the manuscript. MB and XW were involved in data analysis, interpretation of results, and critically reviewing or revising the manuscript for important intellectual content. LDG was involved in the design of the study and critically reviewing or revising the manuscript. MVL was involved in the design of the study, data acquisition, and critically reviewing or revising the manuscript. AGZ was involved in data acquisition and interpretation of results. TN was involved in the design of the study, data analysis, interpretation of results, and critically reviewing or revising the manuscript. ES was involved in the conception and design of the study, interpretation of results, drafting the manuscript, and critically reviewing or revising the manuscript for important intellectual content. SI and ES are the guarantors. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

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Ethical approval: The study was approved by the Austin Hospital Human Research Ethics committee (project number 04958).

Data sharing: The dataset is available from the corresponding author.

The lead and senior authors affirm that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned and registered have been explained.

Dissemination to participants and related patient and public communities: Once published, a national (and international) promotional strategy will be implemented using mainstream and social media. Training for food service staff to implement the methods used in the trial is planned. Outcomes from the trial will be used to improve policy and good clinical practice. Participants and participating facilities will be provided with plain language statements and copies of the publication.

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Web appendix: Supplementary materials
Web appendix: Original and final protocols